Modern Concepts of Cardiovascular Disease

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THE CLINICAL SIGNIFICANCE OF PHYSIOLOGICAL STUDIES IN CARDIAC DECOMPENSATION IN MAN

PART I

Application of physiological methods to the study of congestive failure has resulted in the accumulation of data which have defined the nature of the physiological abnormalities occurring in heart disease, given insight into the mechanisms responsible for the appearance of signs and symptoms and made possible more accurate evaluation of therapy.

Studies of *cardiac output* are of primary importance in evaluating the relation of physiological abnormalities to the clinical manifestations of congestive failure. Two criticisms of this data exist: (1) the accuracy of most methods used is impaired by congestion of the lungs, and (2) the bulk of data has been obtained in patients at rest, in which state symptoms are frequently minimal or absent. The accuracy of data obtained during exercise has not been established. Nevertheless almost all observers, using widely different methods, agree that the cardiac output in patients with cardiac decompensation is low either in the absolute sense or relative to the metabolic needs of the body and the venous pressure. The diminished cardiac output is due mainly to myocardial weakness, but tachycardia and auricular fibrillation may be contributory factors. There is, however, no direct relationship between the severity of the clinical manifestations and the level of the cardiac output. This arises from the fact that the clinical manifestations are due in varying degree to factors other than lowered cardiac output.

A cardiac output which is low relative to the metabolic needs of the body causes tissue anoxia primarily; simultaneously, backing up of blood on the venous side of the circulation causes increased venous pressure. These two abnormalities reflecting both "forward" and "backward" failure are present throughout every organ and tissue, and cause the signs and symptoms of cardiac decompensation. Secondary disturbances of organic function initiated by the low cardiac output and venous stasis assume varying degrees of importance in the genesis of given manifestations of heart failure in different patients. Indeed in some instances these secondary changes may overshadow the low cardiac output and high venous pressure in causing symptoms. Therapy is therefore often properly directed toward correcting these secondary changes, rather than the low cardiac output itself.

The lungs show striking abnormalities of function. Stasis with encroachment on the alveoli by engorged capillaries causes a decrease in lung capacity, distensibility, and elasticity. This is reflected in the vital capacity, which is easily measured and is therefore widely used as a measure of pulmonary congestion in heart disease. However, it must be remembered that pleural effusion, pulmonary fibrosis and emphysema and marked weakness may also be responsible for a low vital capacity. Impaired elasticity and distensibility of the lungs gives rise to a decrease in the negativity of the intrapleural pressure, a factor making for inefficient respiration. The residual air is increased absolutely, or at least relative to the total lung volume, in pulmonary congestion so that heightened respiratory activity is necessary for gaseous interchange. It is apparent that pulmonary function is impaired in a number of different ways, the net result of which is to make increased respiratory activity necessary.

Pulmonary congestion also activates reflexes which give rise to rapid respiration and possibly dyspnea. One of these is the Hering-Breuer reflex, activated by the above-noted impaired collapsibility of the lungs. Another is that first described by Churchill and Cope, which is initiated by increased pressure in the pulmonary vessels. However, evaluation of the role of reflexes in the genesis of the dyspnea of chronic congestive failure is not yet possible, although available evidence suggests their importance in acute pulmonary edema.

The velocity of blood flow is always slowed in congestive failure. Pulmonary congestion, by lengthening and increasing the number of available pathways through the lungs, causes prolongation of circulation time. Decreased cardiac output also results in slowing of flow through the lungs. Estimation of the circulation time is more readily performed than measurement of the cardiac output. Moreover in some patients the cardiac output, though low, is still within normal limits; in such instances the pulmonary blood flow is significantly slowed. The test is of value in distinguishing dyspnea of heart disease from that of pulmonary disease since in the latter normal values are found. It must be borne in mind, however, that slow circulation times are also found in polycythemia, myxedema, and shock.

The arterial blood oxygen saturation is diminished in congestive failure, due mainly to impairment of pulmonary function. Considerably greater decreases in venous blood oxygen saturation occur as a result of the additional factor of peripheral stasis. These changes in blood oxygen content explain in large part the cyanosis of heart disease. Direct measurements of the oxygen tension in the tissues are scanty, but indirect evidence, based on studies of the blood oxygen saturation and also on observations of the favorable results of oxygen therapy, indicate that it is low. Anoxemia, due both to diminished arterial oxygen saturation and to peripheral stasis, is present in all tissues including the respiratory center, and is important in the causation of the dyspnea and increased respiratory activity. Edema formation also depends in part on the presence of chronic anoxemia since the permeability of the capillaries is increased by oxygen lack; lymphatic function may also be impaired.

Carbon dioxide acidosis is recognized as a cause of dyspnea in general, but studies in uncomplicated congestive failure prove that it is not a factor in this condition. Increased respiratory activity in cardiac decompensation results in the blowing off of carbon dioxide so that actually slight shifts toward alkalinity may be found in the arterial blood; changes in arterial blood carbon dioxide content are therefore the effect rather than the cause of hyperventilation.

Elevated blood lactic acid levels are usually found in cardiac decompensation and are evidence of tissue anoxia since they may be reduced by inhalation of high concentrations of oxygen. Anoxia of the skeletal musculature is largely responsible for the elevated blood lactic acid levels although impaired liver function, also due to anoxia, plays a part. The relation of the impaired lactic acid metabolism of congestive failure to tissue anoxia is further shown by the abnormally high and prolonged rises in blood lactic acid following exercise in cardiac patients. Paralleling these abnormal increases in blood lactic acid in such patients after effort are correspondingly great and prolonged oxygen debts. This fact has been made the basis of tests of cardiac function; these though interesting and instructive, are not widely applicable clinically because of technical difficulties and also the wide normal variations. There is evidence pointing to a state of chronic oxygen debt in some patients with cardiac decompensation. The observed abnormalities in lactic acid metabolism not only indicate widespread tissue anoxia but also have important implications in regard to the genesis of dyspnea. The lactic acid metabolism of the brain is important in the regulation of respiratory activity; abnormalities of the former may give rise to dyspnea and increased respiratory activity. In addition, after exercise, a significant degree of acidosis may result from the accumulation of lactic acid in the blood and so give rise to dyspnea. The very high lactic acid levels found in severely decompensated cardiac patients after exercise are important in another respect: maintenance of high concentrations of lactic acid concentrations poisons the heart and thereby gives rise to further impairment of cardiac function.

Venous engorgement is commonly observed in congestive failure and is reflected in the high venous pressure levels found in the veins. These elevated pressures are due largely to the failure of normal emptying of the venous blood into the heart. However, hyperpnea also raises the venous pressure by increasing intrapleural pressure, thereby impeding the entrance of blood into the thorax. The previously described condition of decreased elasticity and collapsibility of the lungs due to pulmonary congestion also raises the intrapleural pressure and contributes to the high venous pressure. It is clear, therefore, that it is not valid to consider an elevated venous pressure as due solely to failure of the right ventricle. There is no constant relationship between the severity of the manifestations of cardiac decompensation and the level of venous pressure. Not infrequently an elevated venous pressure is found weeks or months before symptoms appear; and, on the other hand, marked signs of failure, including edema, may be present without elevation of venous pressure. It also should be borne in mind that an elevated venous pressure occurs in diseases other than cardiac decompensation including marked pulmonary emphysema, pneumothorax, pleural and pericardial effusion, adhesive pericarditis, and mediastinal tumors which interfere with the venous return. The venous pressure may be very high in tricuspid valvular disease and in such instances is not necessarily evidence of severe congestive failure. When the venous pressure is elevated in cardiac decompensation it falls to or toward normal during recovery; failure of an elevated venous pressure to fall, or a continuous rise in venous pressure, indicates a poor prognosis. Elevated venous pressure is of importance in the genesis of edema, although not the sole cause for its appearance, and has also been implicated as a causative factor in orthopnea.

The cerebrospinal fluid pressure is frequently increased in cardiac decompensation since it must always be higher than the venous pressure. Attempts have been made to relate orthopnea to the high spinal fluid pressure of congestive failure but the relationship is not clear although it has been demonstrated that orthopneic patients may show striking amelioration of this symptom for several hours after withdrawal of spinal fluid.

A chemical finding frequently neglected by clinicians is the *low plasma protein level* commonly found in congestive failure. Malnutrition is the most important factor in the production of these low plasma protein levels; other factors include constant albuminuria, loss of protein into the tissues as edema is formed, and removal of large amounts of protein by means of abdominal or thoracic paracenteses. Unless a patient is made acutely uncomfortable by a large amount of fluid in the abdomen or chest, it is preferable to permit him to attempt to re-absorb it, thereby retaining within the body appreciable amounts of protein. Similarly, it appears that low-calory, low-protein diets should not be used for more than a few days in the treatment of cardiac decompensation. Low plasma protein levels are associated with the presence of edema, but a high degree of correlation between the two does not exist. Although the levels found in uncomplicated congestive failure are not as low as in the nephrotic syndrome the decreases are an important factor in the genesis of edema.

The protein content of edema fluid varies greatly. Subcutaneous fluid usually contains less than 0.6 gm. per cent although levels above 1.0 gm. per cent may occur. Chest fluids contain two to four times as much and abdominal fluids even more, levels above 5.0 gm. per cent occurring occasionally in the latter. The protein content of an abdominal or chest fluid therefore cannot by itself be used to differentiate a transudate from an exudate.

(To be continued)

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